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Cardiac output and urea kinetics in dialysis patients: Evidence supporting the regional blood flow model

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Cardiac output and urea kinetics in dialysis patients: Evidence supporting the regional blood flow model. The regional blood flow model predicts that urea sequestration occurs in organs rather than cells, and that post-dialysis urea rebound is a function of both cardiac index (CI) and regional blood flow distribution to muscle. We measured cardiac output (CO) in 100 randomly selected dialysis patients using bioelectric impedance three times during a single dialysis. Mean CO was 5.8 ± 2.1 liter/min and CI averaged 3.1 ± 1.1 liter/min/M^2. CI was negatively correlated with age ($r = -0.48$, $P < 0.01$). CI was strongly affected by vasodilator ingestion (yes, $N = 36$, CI = 3.5 ± 1.2; no, $N = 64$, CI = 2.88 ± 0.92, $P < 0.006$). CI was not associated with systolic, diastolic, or mean blood pressures, nor with Hct, although very few severely anemic patients were in the cohort. Repeat intra-dialytic CO measurements two to three months later in 15 patients with low CI (2.59 ± 0.59 liter/min/M^2) and in 13 patients with high CI (5.00 ± 0.9, $P < 0.001$) during a urea kinetic modeling session including 30 minutes post-dialysis rebound, sampling showed highly reproducible values for CO, with a mean absolute value % difference between CO values measured several months apart of 9.0 ± 17%, $r = 0.92$. Urea rebound expressed as the difference ($\Delta Kt/V_{30}$) between equilibrated and single-pool Kt/V was lower in the high CI group (−0.099 ± 0.07) than in the low CI group (−0.16 ± 0.06, $P = 0.026$), and $\Delta KT/V_{30}$ as well as $\Delta Kt/V_{30}$ divided by K/V correlated with CI ($r = 0.48$ and 0.48, respectively, $P < 0.01$). The RBF model was used to compute a group mean predicted $\Delta Kt/V_{30}$ for the low CI and high CI groups based on measured group mean values for CI and K/V. The predicted $\Delta Kt/V_{30}$ values for the high CI group (−0.097) and the low CI group (−0.183) agreed closely with measured values. RBF modeled values of CO (7.46 ± 2.96 liter/min) were not significantly different from impedance-derived CO (6.93 ± 2.70 liter/min), and the two CO measures correlated significantly ($r = 0.63$, $P = 0.0003$). The results provide support for the regional blood flow model of urea kinetics.

In the single pool urea kinetic model [1], urea within the body is assumed to occupy a single space, the urea concentration of which is in equilibrium with the urea concentration of the mixed venous blood. If urea is removed from only a single body pool, there should be no abrupt increase in the arterial BUN (measured at the vascular access site) beyond that occurring in the first two minutes due to dissipation of the A-V gradient [2]. There should be a slight increase in BUN over time due to continued generation of urea by the body, but this increase should amount to only about 1 mg/dl per hour. Because a much larger post-dialysis urea rebound is observed, especially when a highly efficient dialysis treatment has been given, multicompartment models of urea kinetics have been proposed [3–6]. The simplest such models assume that urea is sequestered within cells during dialysis, and that urea in the extracellular space is in rapid equilibrium with the blood. In this extracellular/intracellular model, the intracellular water is thought to be poorly dialyzed with respect to the extracellular water. As a result, at the end of dialysis, intracellular urea levels would be higher than those in the extracellular space, and rebound would occur due to movement of urea from cells to the extracellular water after dialysis [3–6]. An alternative model is the regional blood flow model, which assumes that urea sequestration during dialysis occurs not within cells, but rather within those organs in which the ratio of blood flow to urea content is low. This group of organs is made up predominantly of muscle, and receives only 15 to 20% of the cardiac outflow, whereas it contains up to 80% of the total body urea. According to the regional blood flow model, muscle tissue is poorly dialyzed, such that at the end of dialysis muscle urea concentration exceeds that of better perfused organs such as the abdominal viscera. In this model, post-dialysis urea rebound is a result of urea movement from muscle to better perfused organs [7–9].

The regional blood flow model predicts that post-dialysis urea rebound will be a function of cardiac index (CI) and the flow fraction to the low-flow compartment fQl (the low-flow compartment is made up mostly of muscle, and possibly also skin and bone). Based on analysis of randomly generated input parameters, we have derived coefficients describing the relation between post-dialysis urea rebound expressed as $\Delta Kt/V$ and the rate of dialysis K/V, expressed as single-pool Kt/V divided by t. For example, if fQl = 0.15 and CI = 2.85, the equations predict that: $\Delta Kt/V = -0.6 \times K/V + 0.03$. The slope term of the above equation (−0.6) is function of CI and fQl [9].

Although the regional blood flow model is grounded in physiology and predicts observed rebound amounts based on reasonable physiologic values for CI and fQl, a more stringent test of the model would be to observe post-dialysis urea rebound in patients in whom CI has been measured, and compare rebound in patients with low and high values for CI. Accordingly, the purposes of the present study were to provide such a test.

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Methods

Patient selection

In Phase 1, after informed consent was obtained according to the principles of the Declaration of Helsinki, CI was measured three times during dialysis in 100 randomly selected maintenance hemodialysis patients being dialyzed three times a week at a large outpatient unit. In Phase 2, CI was remeasured during dialysis in the 15 patients with the highest CI, and the 15 patients with the lowest CI. Patients with upper extremity AV accesses only were studied during Phase 2, to standardize the effect of cardiopulmonary recirculation. Urea modeling was done concurrently with the CI measurements in Phase 2.

Measurements

Cardiac output was measured by thoracic bioelectric impedance (NCCOM3-R7; Bomed, Inc., Irvine, CA, USA) [10, 11]. Predialysis serum urea nitrogen (BUN) levels were taken from arterial needle tubing before injecting heparin. Additional samples were taken one hour into dialysis: full flow inlet, full flow outlet, and 15 to 20 seconds slow flow inlet. At the end of dialysis, samples were taken as follows: full flow inlet, 15 to 20 seconds slow flow inlet, two minutes slow flow inlet, and 30 minutes post-dialysis.

Computations

Anthropometrics. Surface area was estimated from the DuBois equation [12]. The anthropometric urea distribution volume V was obtained from the surface area by the Hume-Weyers regression [13].

Urea kinetic parameters. Single pool Kt/V was computed according to the second generation Daugirdas equation using the anthropometric V instead of W [14]:

$$= -\ln(R - 0.008t) + (4 - 3.5R) \times 0.55 \times UF/V$$

where R = 15 to 20 seconds BUNpost divided by BUNpre, V = anthropometric V, t = dialysis session length (hrs), and UF = wt loss in kg. Equilibrated Kt/V was computed using the same formula, but with R = 30 minutes postBUN/preBUN. ΔKt/V was then calculated as equilibrated Kt/V − single-pool Kt/V. Access recirculation was measured in all patients near the end of dialysis using ultrasound dilution as described by Krivitski [15] and was zero in all but one patient (in the low CI group) whose data were excluded from the analysis.

Regional blood flow equations

As in Daugirdas and Schneditz [9], random input values were used to generate terms for the general equation:

$$\Delta Kt/V = -m \times K/V + b$$

The group mean cardiac index for the low CI group or for the high CI group was put into the equation with random assignment of the remaining parameters to solve for the optimum coefficients "m" and "b". In this manner, two different sets of coefficients were established for each group. These were then used to solve for the expected group mean ΔKt/V for the low and high CI groups, respectively. The equations used and the parameters were those listed in the Appendix of Daugirdas and Schneditz [9]. There are three typographical errors in that appendix: (1) In the formula for auch, line 2, there should be no parenthesis prior to la1-q; (2) the parenthesis prior to a21 in the formula for a22 should be deleted; and (3) in the formula for doseh, g should be gn. Otherwise, the only parameters changed were the use of a constant 0.85 for the fQh term, and use of either 2.6 or 5.0 for the CI term. The coefficients "m" and "b" were obtained by regressing $\Delta Kt/V_{30}$ (the difference between the equilibrated Kt/V and that at the immediate end of dialysis) on K/V (computed by dividing the immediate post-dialysis Kt/V by the hours of dialysis.

Modeling of cardiac output for individual patients using the regional blood flow model

Two model parameters, cardiac output (CO) and urea distribution volume (V) were identified by fitting experimental data to the regional blood flow model [7–9]. Pre-dialysis, end-dialysis and 30-minute post-dialysis BUN values, ultrafiltration rates, blood-side urea clearances (K), treatment times (t), extracorporeal blood flows (Qb), and urea generation rates (G, estimated from BUNpost and BUNpre and corrected for 93% plasma water content) were used as model inputs, while CO, V, equilibrated KtV (KtVeq), and SDs (SD) between residuals of experimental and fitted data were obtained as model outputs.

Data fitting was done using the Solver option offered by MS Excel version 5.0, minimizing SD with constraints for cardiac output (CO: $0.5 \times 2.4 \times$ body surface area $< CO < 1.5 \times 4.2 \times$ body surface area), V ($0.5 \times TBW_{anthropometric} < V < 1.5 \times TBW_{anthropometric}$), BUNend ($-0.01 <$ (BUNend, exp-BUNend, model)/BUNpre < 0.01), and 30 minutes BUNpost ($-0.02 <$ (BUNpost30, exp-BUNpost30, model)/BUNpre < 0.02). Model parameters such as access flow (Qac = 0.8 liter/min), fractional volume of the high flow compartment (fVh = 0.2), fractional perfusion of the high flow compartment (fQh = 0.85), and capillary permeability surface area product (PS = 20 liter/min) were assumed at constant values as reported previously. Access recirculation (Ra = 0) and residual clearance (Kr = 0) were assumed to be negligible.

Effective clearances (Cl_{high}, Cl_{low}) were calculated from analytical expressions of areas under modeled concentration curves (AUC) and modeled amounts of BUN removed from both high (doseh) and low flow compartments (dosel). Equilibrated KtV (KtVeq) was calculated as the sum of high and low flow compartment clearances, experimental treatment times, and modeled end-dialytic BUN-distribution volumes as described previously [7–9].

Results

Impedance cardiac output in Phase 1 (100 dialysis patients)

Mean CO was 5.8 ± 2.1 liter/min, and mean CI was 3.1 ± 1.1 liter/min/M^2. Systolic, diastolic, and mean blood pressures averaged 139, 79, and 99 mm Hg. Mean body surface area (SA) was 1.86 ± 0.25 M^2, Anthropometric V was 39.5 ± 7.8 L. Mean Hct was 32% ± 5.2. Half of the patients were taking anti-hypertensive medications and 36% were on vasodilators (minoxidil, hydralazine). Cardiac index was negatively correlated with age (Fig. 1, $r = -0.48$, $P < 0.01$). CI was strongly affected by vasodilator ingestion (yes, $N = 36$, CI = 3.5 ± 1.2; no, $N = 64$, CI = 2.88 ± 0.92, $P < 0.006$). Frequency distribution plots of CI in patients taking and not taking vasodilators are shown in Figure 2. CI measurements were highly reproducible over time. The coefficient of variation of intradialytic CI measurements repeated within

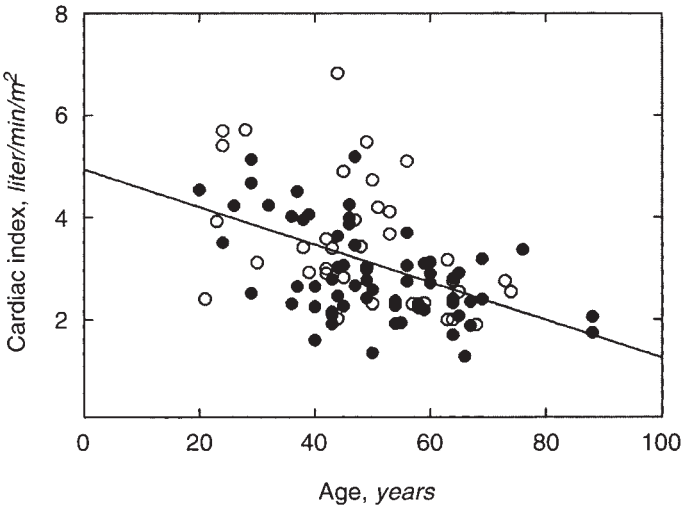

Fig. 1. *Relation between measured intradialytic CI and patient age.* The patients are grouped into those taking vasodilators (○) and those not taking such medications (●). The negative correlation coefficient is highly significant, and the negative relation between age and CI has been observed by others ($r = -0.48$).

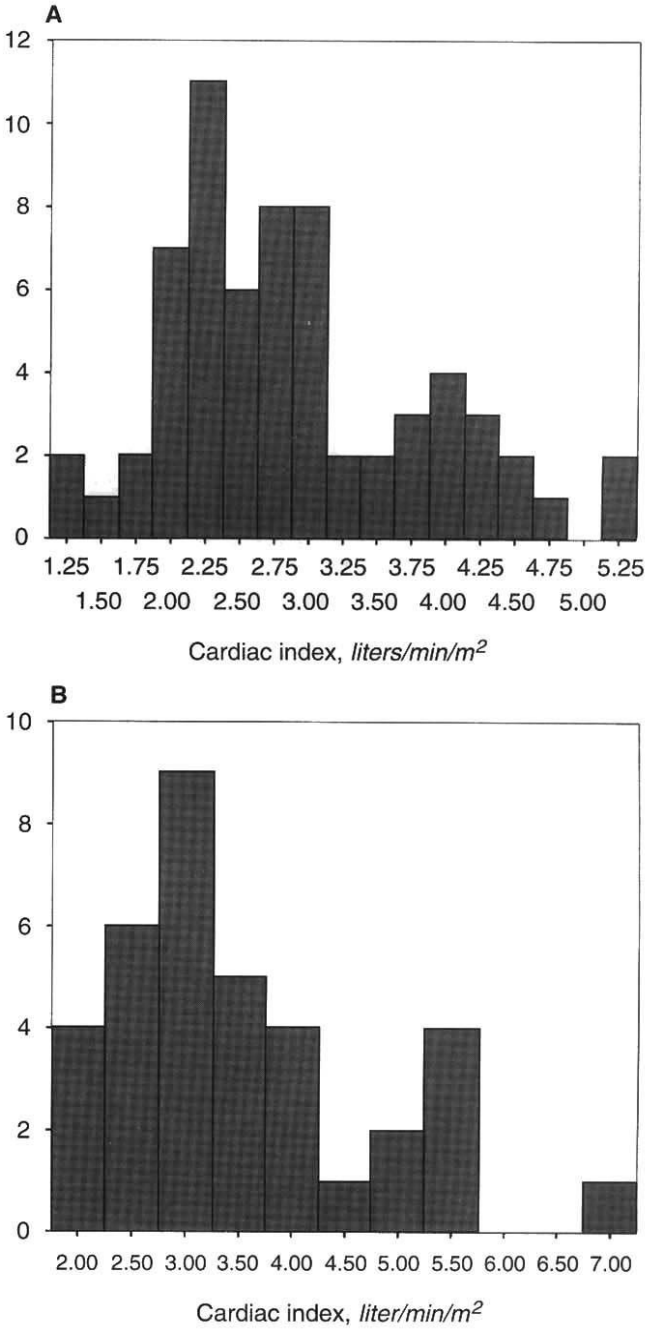

Fig. 2. A. *Frequency distribution plots of CI in patients not taking vasodilators.* The mean value is 2.88 liter/min/M^2; SD = 0.92; $N = 64$. **B.** Frequency distribution plots of CI in patients taking vasodilators. The mean value is 3.51 liter/min/M^2, significantly higher than in patients not on this type of medication. The SD is 1.26; $N = 36$.

several hours was quite small (5.2 ± 3.9%). A sex effect (63 males, 37 females) was present for CO but not for CI. Twenty-five percent of the patients were obese by body mass index (BMI); CI (but not CO) was reduced in obese patients, suggesting perhaps deviation from an/thropometric assumptions in obese patients. CI was not associated with systolic, diastolic, or mean blood pressures, nor with Hct. Of the 100 patients, 15 had a venovenous access, 80 had a lower arm graft or fistula, and 5 had an upper arm or thigh graft. CI did not differ significantly among these three groups (2.8 ± 0.79, 3.1 ± 1.1, and 3.7 ± 1.0 liter/min/M^2, respectively).

Impedance cardiac output and urea kinetic modeling result (Phase 2)

In addition to the patient with access recirculation, data from one additional patient with low CI were excluded due to several missing samples. There remained data on 15 patients in the high CI group and on 13 patients in the low CI group. The main modeling parameters are listed in Table 1 for the high and low CI groups. In the repeat measurement of CI in these 30 patients with the lowest and highest CI values, which were taken two to three months later, the mean absolute value % difference between intradialytic CO values at repeat evaluation versus CO values at initial screening was 9.0 ± 17%, $r = 0.92$. Thus, the separation in CI between the two groups remained high, with almost a 100% mean difference between them (Table 1). There was no difference in weight, sex ratio, or treatment parameters between the two groups, although the high CI group tended to have more women and weigh less. The measured dialyzer clearance, modeled single pool volume, and rate of dialysis (K/V) were all quite similar in the two groups. Despite this, there was a marked difference in ΔKt/V between the two groups, averaging −0.16 Kt/V units in the low CI group and −0.099 in the high CI group. The mean ΔKt/V values were quite similar to those predicted (−0.183 and −0.097, respectively) on the basis of the regional blood flow model calculations (Fig. 3). When ΔKt/V was plotted against CI, a highly significant correlation ($r = 0.48$, $P < 0.01$) was found (Fig. 4).

The two-minute post-dialysis samples also were used to examine rebound. The $\Delta Kt/V_{2\text{-}30}$ (defined as equilibrated Kt/V − Kt/V computed using the Daugirdas second generation formula and the post-samples obtained after 2 min of slow flow) averaged −0.12 ± 0.062 in the low CI group, and −0.074 ± 0.069 in the high CI group ($P < 0.05$). The $\Delta Kt/V_{2\text{-}30}$ value correlated with the

Table 1. Modeling results (mean ± SD)

	Low cardiac index	High cardiac index	*P*
Number of patients	13	15	
Impedance CI *liter/min/M²*	2.6 ± 0.59	5.0 ± 0.93	<0.001
Impedance CO *liter/min*	4.6 ± 1.15	8.9 ± 1.9	<0.001
RBF modeled CO *liter/min*	5.9 ± 2.7	8.8 ± 2.5	<0.001
Weight *kg*	75.4 ± 12.6	66.3 ± 11.5	NS
Sex (M:F)	6:7	4:11	NS
Session length *hours*	3.7 ± 0.38	3.5 ± 0.42	NS
Dialyzer blood water K_d *ml/min*	180 ± 24	185 ± 27	NS
Qb *ml/min*	342 ± 19	346 ± 13	NS
Predialysis BUN *mg/dl*	49 ± 9.2	48 ± 18	NS
Postdialysis BUN *mg/dl*	29 ± 5.6	29 ± 11	NS
$PDUR_p$ *% of post-sample*	5.7 ± 2.3	3.7 ± 2.6	<0.05
Kt/Vsp	1.14 ± 0.15	1.10 ± 0.21	NS
Kt/Veq	0.98 ± 0.13	1.00 ± 0.21	NS
K/V	0.31 ± 0.05	0.32 ± 0.07	NS
ΔKt/V30-measured	−0.16 ± 0.06	−0.099 ± 0.07	<0.026
Predicted group K/V slope ("m")	−0.69	−0.32	
Predicted group intercept ("b")	0.03	0.005	
ΔKt/V30-rbf. predicted for the group	−0.183	−0.097	NA
Modeled single-pool V (V_{sp})	35.7 ± 8.0	36.2 ± 8.3	NS
Anthropometric V (V_a)	37.6 ± 4.0	35.4 ± 3.7	NS
Vratio (V_{sp}/V_a)	0.96 ± 0.24	1.01 ± 0.15	NS

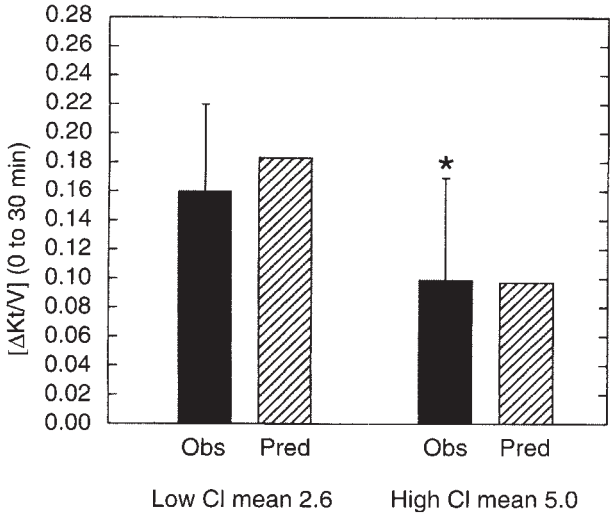

Fig. 3. *Bar graph showing the CI in the low and high CI groups as well as the mean observed and RBF-predicted ΔKt/V values in each group.* The ΔKt/V values are significantly different and the means are similar to the predicted group means based on the RBF model.

impedance derived CI value ($r = 0.46$, $P < 0.05$). Because it is known that the A/V urea gradient will be increased in patients with low CI [2], these results suggest that CI values affect urea rebound even after the A/V urea gradient closes, and point to an effect of CI on the true "compartment" phase of the urea rebound.

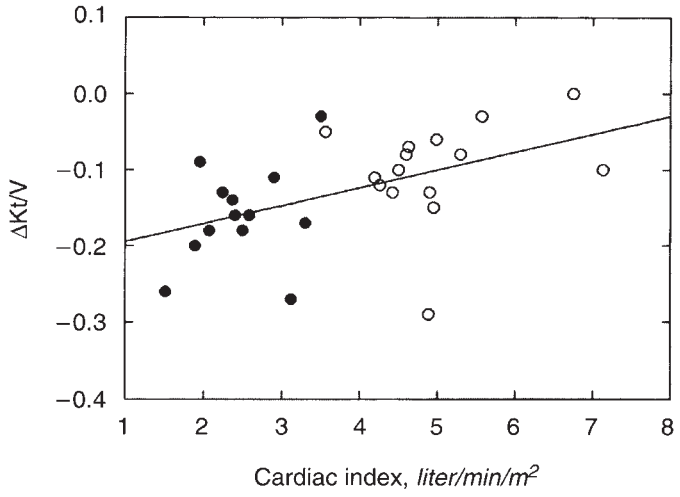

Fig. 4. *Regression plot of ΔKt/V against CI in patients preselected for high (N = 15) and low (N = 13) CI.* The regression is highly significant ($P < 0.01$). Symbols are: (●) patients initially drawn from the low CI group; (○) patients from the high CI group; $r = 0.48$.

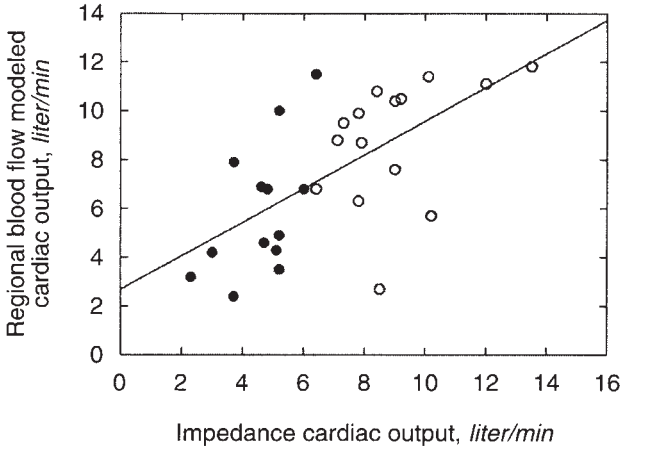

Fig. 5. *Regression plot of regional blood flow modeled cardiac output versus impedance measured cardiac output.* The regression coefficient ($r = 0.63$) is highly significant ($P < 0.001$). Symbols are: (●) low CO group; (○) high CO group.

Discussion

Our results demonstrate that intradialytic cardiac index (CI) in dialysis patients not taking vasodilators tends to be rather low, about 2.9 liter/min/M^2. This is similar to results found by authors of the present paper using either thoracic electric bioimpedance [16–18] or thermodilution [19]. In the 100 patients that were screened, an inverse relationship between cardiac output and age was demonstrated. Patients taking hydralazine or minoxidil tended to have higher CI values than those not taking anti-hypertensive medications or who were taking other anti-hypertensive medications. The type of vascular access did not affect the CI, although few patients ($N = 5$) with upper arm or thigh grafts were included in the analysis. There was a non-significant trend for CI to be higher in patients with lower arm AV accesses (mean CI = 3.13) than with venovenous accesses (mean CI = 2.79).

Our data suggest that the thoracic electric bioimpedance (TEB) method of measuring cardiac output [10, 11] in dialysis patients

has a low coefficient of variation on repeat assessment. The low coefficient of variation of 5% within a single dialysis and 9% across means of two intradialytic CI measurements taken three months apart is quite impressive. This is now the fourth published study in which we used TEB to measure CO in dialysis patients. Initially, we found that TEB-CO measurements identified the well known increase in CI during acetate-based dialysate use [16]. In a second study, we found that food ingestion during dialysis was associated with an accelerated fall in MAP but a tendency toward a rise in CI [17], consistent with published results of others [20]. We also found that lowering dialysate temperature increased blood pressure by maintaining or increasing peripheral vascular resistance [18]. In abstract form, we recently have examined the ability of TEB-CO to predict dialysis-induced hypotension, and found that TEB-CO fell in all but one patient to similar levels during such episodes [21]. Taken together, this experience suggests that TEB-CO is a useful tool to measure CI in dialysis patients. The hemodynamic relevance of TEB-CO measurements is further enhanced by the relations between TEB-CI and urea rebound identified in the present study.

The regional blood flow model of urea kinetics predicts that whenever muscle blood flow and cardiac output are low, post-dialysis rebound will be large. Conversely, when muscle blood flow and cardiac index are high, rebound should be small. The exact solution to the RBF model is now available [8] and allows for quantitative predictions of the relationship between CI and postdialysis urea rebound. We have used this model and the knowledge from observations by us and others that mean CI is often about 2.85, mean access blood flow about 800 ml/min, and the physiological inference that perfusion to the low flow organs is about 15% of cardiac output, to define a formula to predict the post-dialysis rebound in the majority of patients [9], a formula that has been found to be useful by others as well [22]. The present results suggest that, in the absence of vasodilator ingestion, mean intradialytic CI is indeed about 2.9, and further support the appropriateness of the rate equation [9] to predict $\Delta Kt/V$. The present results also demonstrate that, if we preselect patients for low or high CI, then the post-dialysis rebound will differ in direction and magnitude as predicted by the RBF model. Furthermore, cardiac output as predicted by the regional blood flow model, making an assumption that 15% of cardiac output goes to the low flow fraction, and that access recirculation is about 800 ml/min, is not significantly different from that measured by impedance. These results further support both the regional blood flow model and the usefulness of thoracic bioelectric impedance in dialysis patients.

In summary, thoracic electric bioimpedance measurement of cardiac output (CO) in dialysis patients is highly reproducible. Cardiac output correlates negatively with age. Cardiac index averaged 2.9 in patients not taking vasodilators, close to the mean value of 2.85 used in deriving a prediction equation of post-dialysis urea rebound based on the regional blood flow model [9]. Cardiac output was significantly higher in patients receiving vasodilators (hydralazine, minoxidil). Post-dialysis urea rebound correlated with intradialytic CI and was different in patients with high cardiac index versus patients with low cardiac index. The relation between cardiac index and post-dialysis urea rebound was as predicted by the regional blood flow model.

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